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Case Report

A Case of Radiofrequency Catheter Ablation of Ventricular Tachycardia Associated with an Old Myocardial Infarction Guided by a Noncontact Mapping System

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We describe here a 72-year-old man with ventricular tachycardia (VT) associated with an old myocardial infarction, in whom noncontact mapping guided radiofrequency catheter ablation (RFCA) successfully eliminated the VT. Right ventricular pacing induced 3 VTs with different QRS morphologies and axes, 2 of which were hemodynamically unstable. A dynamic virtual activation map constructed during the VTs superimposed on a virtual voltage map constructed during sinus rhythm demonstrated that all VTs shared a single large myocardial scar in the inferolateral portion of the left ventricle which served as a slow conduction zone. All VTs were eliminated by RFCA at the exit or within the critical slow conduction zone. The patient has been free from any VT recurrences during a follow-up period of 22 months.
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Key words: ventricular tachycardia, noncontact mapping system, radiofrequency catheter ablation, activation, myocardial infarction

Introduction

The strategy for radiofrequency catheter ablation (RFCA) of ventricular tachycardia (VT) associated with an old myocardial infarction (OMI) consists of substrate modification, circuit modification, or both. In the former, RFCA is usually performed at the exit site from the slow conduction zone and/or at a narrow isthmus in an attempt to eliminate all potential reentry circuits. The target, however, is an estimated critical reentrant circuit speculated from the substrate map obtained during sinus rhythm or atrial pacing, and thus, there is the possibility that an unexpected reentry circuit may remain after the ablation or an undemonstrated functional slow conduction zone may develop during the VT. In the circuit modification strategy, on the other hand, real reentrant circuits are directly targeted, but that has been limited in use to those with hemodynamically tolerated VT because a time-consuming process for the activation and entrainment mapping is usually required.

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The noncontact mapping system (NCM) allows us to perform a detailed activation and substrate analysis in a beat-to-beat fashion using isopotential maps constructed with virtual unipolar electrograms, which lead to the RFCA of the OMI-VT guided by a combination of the virtual activation map during the tachycardia and virtual substrate map constructed during sinus rhythm. Here we describe a case of an RFCA of OMI-VT using NCM.

Case Report

A 72-year-old man with drug-refractory VT was referred for an electrophysiologic study and RFCA. The patient had a history of an inferolateral MI following coronary artery bypass grafting in 1996, and frequent episodes of monomorphic VT requiring DC shock to terminate it since November 2004. Amiodarone was discontinued due to the occurrence of interstitial pneumonia. Echocardiography revealed an akinetic inferolateral left ventricular wall motion in addition to a global left ventricular wall motion reduction with an ejection fraction of 0.34. The baseline heart rhythm was sinus rhythm with left axis deviation (LAD), abnormal Q waves in the inferior leads and a poor R wave progression in the chest leads.

After written informed consent was given, the electrophysiologic study and RFCA were performed under conscious sedation. One twenty-pole, and two quadripolar electrode catheters were located in the coronary sinus, right ventricular apex (RVA) and His bundle region, respectively, via the femoral veins for contact bipolar recording and pacing. Using a standard Brockenbrough technique, a long transseptal sheath (Mullins, Medtronic, Minneapolis, MN, USA) was introduced into the left ventricle (LV) under fluoroscopic guidance. A multielectrode array catheter (MEA, St. Jude Medical, St. Paul, MN, USA) was placed in the LV via the mitral valve through the long transseptal sheath, and the tip of the MEA was placed in the LV apex. The NCM was used to analyze the activation and substrate information of the LV during the VT and sinus rhythm, in which virtual unipolar electrograms were filtered with a band pass of 2.0–500 Hz, and an isopotential map was constructed with those multiple virtual unipolar electrograms as previously reported.[1)]

A deflectable 7-French quadripolar catheter with an 8-mm distal electrode (Fantasista, Japan Life Line, Japan) was used for the mapping and ablation, which was advanced into the LV via the retrograde transaortic approach, and later via an additional atrial transseptal sheath (SL1, St. Jude Medical, Minnetonka, MN, USA). Intravenous heparin was administered to maintain an activated clotting time (ACT) of >300 seconds immediately after the atrial transseptal puncture. The ACT levels were monitored every 30 minutes, and if they were <300 seconds, additional heparin was injected to maintain an ACT of >300 seconds.

To begin with, the LV geometry was depicted with the NCM (EnSite 3000, version 6.0J, St. Jude Medical, Minnetonka, MN, USA) using a roving quadripolar electrode catheter within the LV during sinus rhythm. A dynamic substrate map (DSM) was obtained during sinus rhythm as reported previously,[2)] in which a low voltage zone was defined as an area with an amplitude of the maximum peak negative value of $<50\%$ among all the virtual unipolar electrograms during sinus rhythm, and markers were placed surrounding the low voltage zone to identify the borders surrounding it. The DSM revealed a localized low voltage zone in the inferolateral portion of the basal LV, which seemed to correspond to the scar due to an inferolateral OMI.

The VT was repeatedly induced by burst pacing from the RVA, which was performed at a stimulus strength of twice the diastolic threshold and with a pulse width of 2 msec using a programmable stimulator (SEC-3102, Nihon Kohden, Japan). The initially induced monomorphic VT had a right bundle branch block (RBBB) pattern and inferior axis with a cycle length of 368 msec (**Figure 1A**, VT#1), and it was immediately terminated by RVA pacing because of hemodynamic instability. The same VT was reproducibly induced and the NCM was used to analyze the VT activation as previously described.[1)] A virtual activation map of the VT superimposed on the DSM map obtained during sinus rhythm revealed the low voltage zone acted as a slow conduction zone with the activation spreading out from the anterolateral middle LV with the low voltage zone to the entire LV (**Figure 2A**). At the exit site demonstrated by the virtual activation map, the virtual unipolar electrograms exhibited a QS morphology with a fast downstroke slope preceding the QRS complex of the VT. After the entire LA was activated, the wavefront narrowed and entered the low voltage zone. The border of the healthy myocardium and the low voltage zone from which the wavefront entered the low voltage zone was considered to be the entrance site. The double potentials with low voltage amplitude were recorded in the contact bipolar electrograms from the ablation catheter at the exit site (**Figure 3A**). The ablation catheter, introduced into the LV by a retrograde

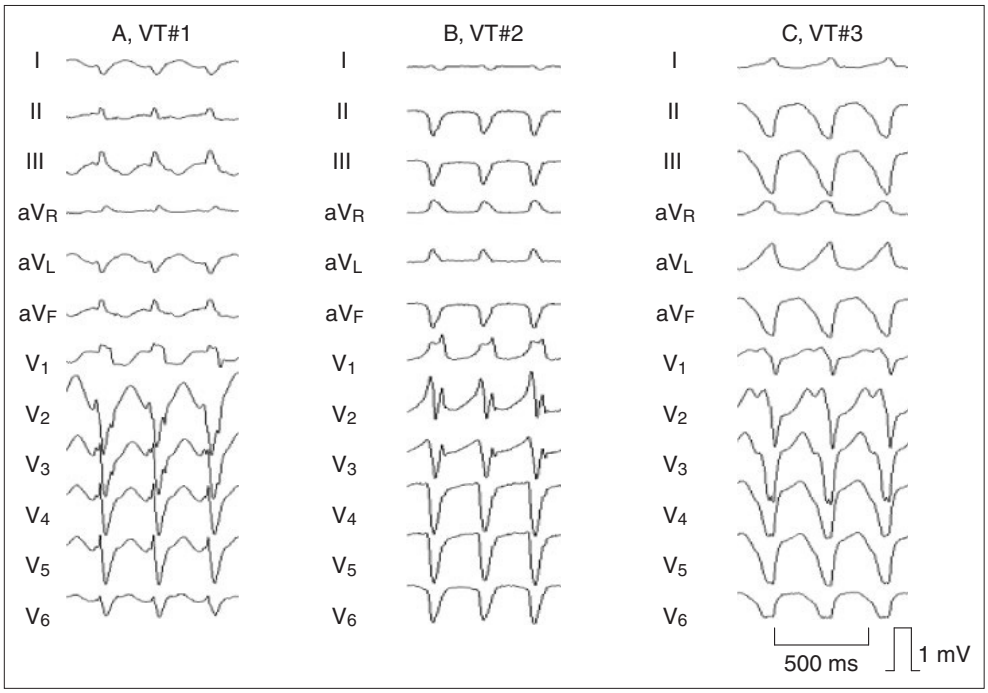

Figure 1
The twelve-lead ECGs of the VTs induced by right ventricular apex pacing: a VT with a right bundle branch block (RBBB) pattern and inferior axis (**A**, VT#1), VT with an RBBB pattern and left axis deviation (LAD) (**B**, VT#2) and VT with a left bundle branch block pattern and LAD (**C**, VT#3).

trans-aortic approach, was easily navigated to the exit site by the NCM and RF energy was applied at that site during sinus rhythm with a target temperature of 55 °C and maximum power output of 50 W. Additional RF energy was delivered at the exit site and the vicinity on the scar border which was defined as 50% of the maximal peak negative value of unipolar electrogram during sinus rhythm.

After that, the VT#1 was no longer inducible, but a second VT with a cycle length of 326 msec, a RBBB QRS pattern and left axis deviation (**Figure 1B**, VT#2) was induced by RV pacing. This VT seemed to be the clinical one due to the similar QRS morphology to that of the clinically documented VT, but had to be terminated by rapid RV pacing because of hemodynamic instability. The regional dynamic activation map constructed with the NCM revealed that the VT activation went through the slow conduction zone with the entrance at the anterolateral LV and the exit at the posterolateral LV (**Figure 2B**). To eliminate this VT, the ablation catheter was introduced into the LV through the mitral valve using the transseptal approach because trying to manipulate the catheter to the exit site using the retrograde trans-aortic approach was difficult. The fractionated potentials with low voltage amplitude were recorded in the contact bipolar electrograms from the ablation catheter at the exit site (**Figure 3B**). After the RF energy applications to the exit and its surrounding sites during sinus rhythm, the VT#2 was no longer inducible by RV pacing.

RV pacing, however, induced a third VT with a cycle length of 385 msec and a left bundle branch block pattern and LAD (**Figure 1C**, VT#3). The ablation of the VT was performed during on-going VT because of its hemodynamic stability. The regional dynamic activation map constructed with the NCM revealed that the VT activation traveled down through the slow conduction zone, in which the entrance was similar to that of the VT#2 but the wavefront proceeding within the slow conduction zone was blocked at the exit of the VT#2, and thus, propagated slowly within the slow conduction zone toward another exit site near the posterior LV (**Figure 2C**). Several RF energy applications were performed at that exit site; however, the VT did not terminate. Therefore, we tried to ablate a more proximal portion of the slow conduction zone where the diastolic pathway was identified by the NCM during the VT#3 and double mid-diastolic potentials preceding the QRS onset by 140 msec (first component) and 40 msec (second component) were recorded in the contact bipolar electrograms from the ablation catheter (**Figure 4**). Entrainment pacing was

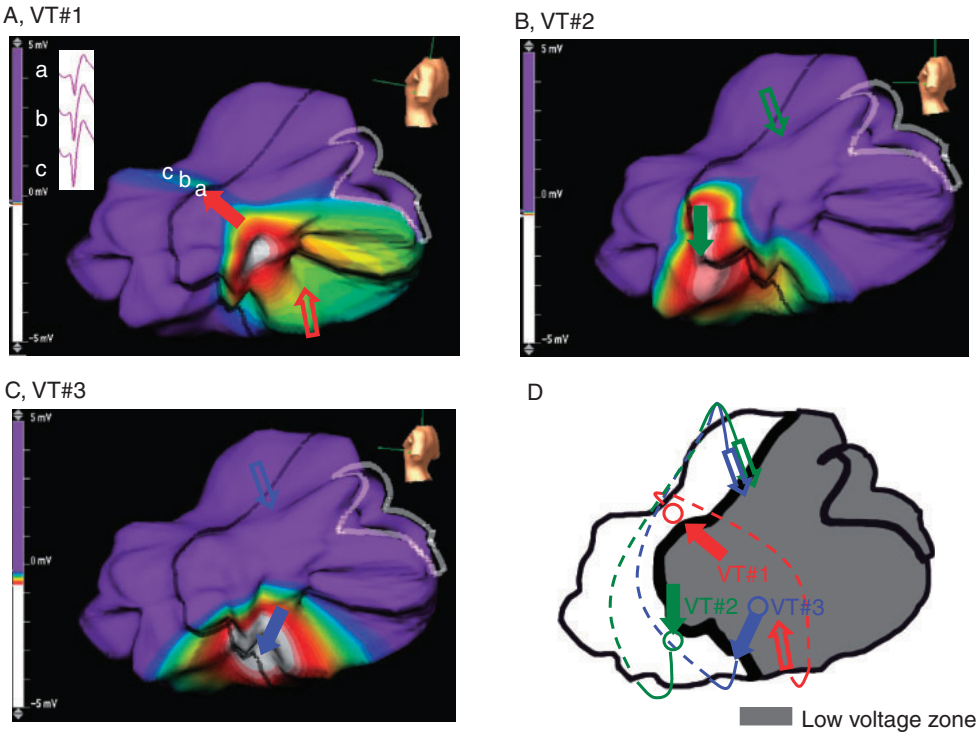

Figure 2 An isopotential map at the QRS onset of VT#1 (**A**), VT#2 (**B**) and VT#3 (**C**). Panels **A** to **D** are obtained in the left lateral view. The left ventricular endocardium is divided into 2 areas by the black line according to the voltage amplitude (**D**). The low voltage zone is the mitral annulus side, and non-low voltage zone is the left ventricular apex side. The white line in panels **A** to **D** stands for the mitral annulus. The closed arrow in each panel indicates the direction of the activation from the low voltage zone to the entire left ventricle (red arrow: VT#1, green arrow: VT#2 and blue arrow: VT#3). The open arrow in each panel indicates the direction of the activation from the LV to the low voltage zone. Virtual unipolar electrograms at site **a**, **b**, and **c** are shown in panel A. Virtual unipolar electrograms at the exit site (**a**) shows QS deflection while the sites away from the exit site (**b** and **c**) have electrograms with R wave in the initial part of the electrogram. (**D**) Diagrammatic representation of the LV with the activation sequence of each VT. The low voltage area at the inferolateral site of the basal LV is described as a gray area. The activation sequence is traced with lines based on the activation wavefront analyzed by the noncontact mapping system. The circles show the sites of the successful radiofrequency catheter ablation of each VT. All the VTs have their exit and entrance portions at the border of the low voltage zone. Note that all the VTs shared a single large myocardial low voltage zone in the inferolateral left ventricle which served as a slow conduction zone. The entrance sites from the LV to the low voltage zone of VT#2 and that of VT#3 are almost the same. LV: left ventricle.

attempted but the site was not locally captured with the maximal pacing out put (9.9 V). An RF energy application was delivered at that site, and the VT#3 terminated during the ablation. Thereafter no VT could be induced by ventricular extrastimuli of up to 3 successive beats or burst pacing of up to 260 beats per minute in addition to an isoproterenol injection.

No procedural complications such as thromboemboli or valve/chordae injuries occurred during or after the ablation procedure. After the RFCA procedure, the patient underwent an ICD implantation to prevent any sudden death due to an unpredictable ventricular tachyarrhythmia occurrence. The patient has been free from any VT recurrences or ICD shocks during a follow-up period of 24 months without any antiarrhythmic medications other than β-blocker.

Discussion

The present case demonstrated the feasibility of OMI-VT ablation guided by VT activation analysis using the NCM. Although 2 of 3 induced VTs were hemodynamically unstable and required termination by RVA pacing immediately after the occurrence, the dynamic VT activation map superimposed on the virtual voltage map constructed during sinus rhythm clearly elucidated the entire reentrant activation of all 3 VTs using the NCM. All 3 VTs were eliminated

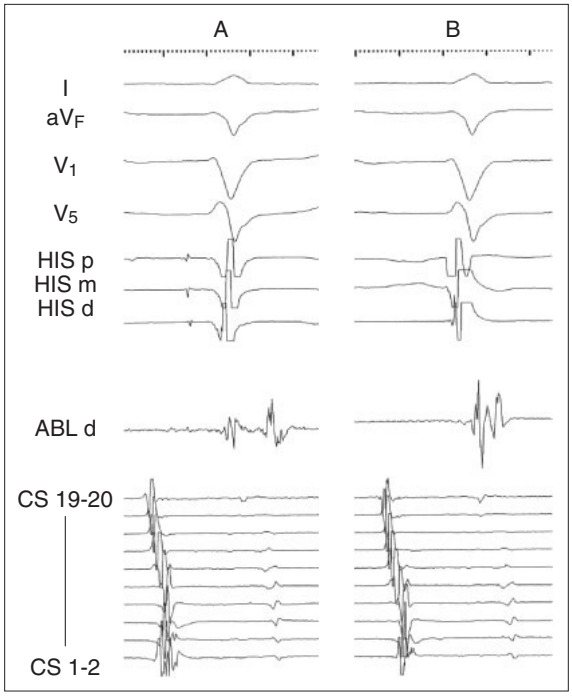

Figure 3 Intracardiac recordings at the exit sites of VT#1 (**A**) and VT#2 (**B**) during sinus rhythm.
The double or fractionated potentials are found at the exit sites. The amplitude of the electrogram at each exit site are 0.31 mV and 0.45 mV, respectively.

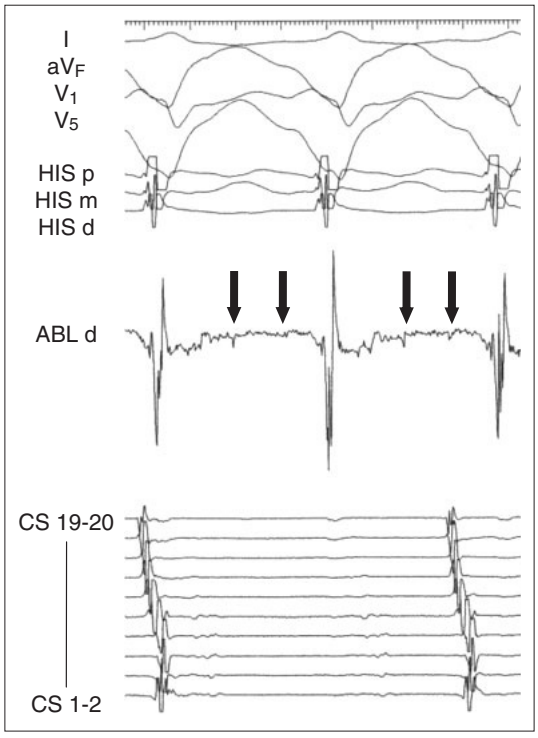

Figure 4 Intracardiac recordings during the VT#3 at the successful ablation site.
In the contact bipolar recording, the amplitude of the electrogram at that site is 0.27 mV, and mid diastolic potentials (closed arrow) preceding the QRS onset by 140 msec (first component) and 40 msec (second component) are recorded.

by RFCA at the exit or within the critical slow conduction zone. VT#3 was not eliminated by the RF energy application to the exit site from the low voltage zone. The exit site of the VT might be located at the more epicardial site compared with other exit sites. The RF energy was applied to the VT exit and/or to a diastolic pathway identified by the system as the ablation strategy using an array mode of the NCM.[1)] Therefore RF ablation was performed at the diastolic pathway during the VT#3 after RF was unsuccessfully applied to the VT exit. The pace mapping of the VT could not be performed at the exit sites of VT#1 and VT#2 because the pacing failed to capture these sites.

The RFCA of OMI-VT was initially developed to ablate the critical reentrant circuit of an on-going VT, where concealed entrainment was shown as well as a similar post pacing interval to the VT cycle length. This approach has been limited for use in the clinical practice because it can not be applied to unmappable VTs including hemodynamically unstable, nonsustained or pleomorphic VTs, but only to hemodynamically stable monomorphic sustained VT because the time-consuming process of activation mapping and the entrainment technique are usually required.

To overcome these limitations, the substrate modification approach has been performed in an attempt to eliminate all potential arrhythmia mechanisms in order to abolish not only hemodynamically stable monomorphic sustained VT, but also all potential VTs including hemodynamically unstable, nonsustained or pleomorphic VTs. This approach consists of several sub-strategies.[3–8)] The first one is to ablate the potential VT reentry circuit identified by pace map at the scar border zone which has been described with contact bipolar electrograms during sinus rhythm. The second one is to destroy the narrow conducting channels between dense scars within the infarct areas which are identified as unexcitable dense scars, or extremely low voltage scars. The last one is to identify the critical pathway within the infarct areas with a delayed potential recorded during sinus rhythm, where a good pace match was obtained during pace mapping. In these strategies, the target is, however, an estimated critical reentrant circuit rather than the real reentrant circuit, and thus, there are some concerns that an unexpected reentry circuit may still remain or an undemonstrated functional slow conduction zone

may develop during the VT when excessive energy applications to by-stander pathways, dead end pathways, or even innocent areas outside the reentry circuit might be performed in this strategy.[3,9)]

The NCM, however, allowed us to perform an activation-guided circuit modification, in which a VT activation analysis was performed on the base of the DSM constructed during sinus rhythm. The map could be constructed for any VT irrespective of the hemodynamic condition, VT duration, or whether it is monomorphic or pleomorphic. The NCM has several potential advantages over conventional mapping techniques, in which a 3-dimentional beat-to-beat activation analysis can be performed with a high resolution of >3000 endocardial points, but some concerns have been raised in terms of the reliability of the virtual unipolar electrograms at remote points of >4.0 cm from the center of the MEA, weak resolution capability of the low frequency/amplitude electrograms, and influence of the repolarization on the interpretation of real potentials. However, recent studies have demonstrated the safety and usefulness of the NCM for the mapping and ablation of VT associated with an OMI,[1,2,10–12)] in which the NCM was considered useful even in patients with hemodynamically unstable and/or nonsustained VTs.[13)]

The significance of the DSM for elucidating the low voltage zones is becoming recognized.[2)] Interestingly, the low voltage zones identified by the DSM maps constructed during sinus rhythm acted as slow conduction zones in all 3 VTs in this case. It was noted that both the entrance and exit from the slow conduction zone corresponded to the low voltage border. That fact confirmed the capability of the DSM map for identifying low voltage zones with slow conduction properties, even though the low voltage was not confirmed by the contact bipolar recordings.

In conclusion, the virtual VT activation map superimposed on the virtual voltage map constructed during sinus rhythm demonstrated the entire reentry circuit in the 3 VTs with variable hemodynamic conditions, and RFCA guided by those isopotential maps eliminated all the VTs. Activation guided RFCA with the array mode of the NCM might be effective and safe to eliminate OMI-VTs.

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